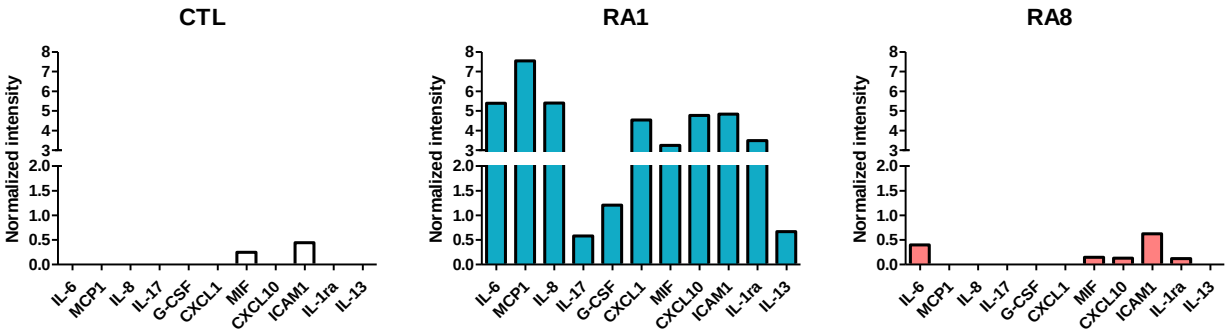

Figure S1. Evaluation of synovial fluids inflammatory status. Graphical representation of Figure 1A. SF were represented depending on their inflammatory status (blue being very pro-inflammatory, red slightly inflammatory). Pro-inflammatory mediators IL-6, MCP1, IL-8, IL-17, G-CSF, MIF, CXCL10, ICAM-1, IL-1RA, IL-13 were identified and semi-quantified using an antibody-based membrane array in CTL, RA1 and RA8 synovial fluids.

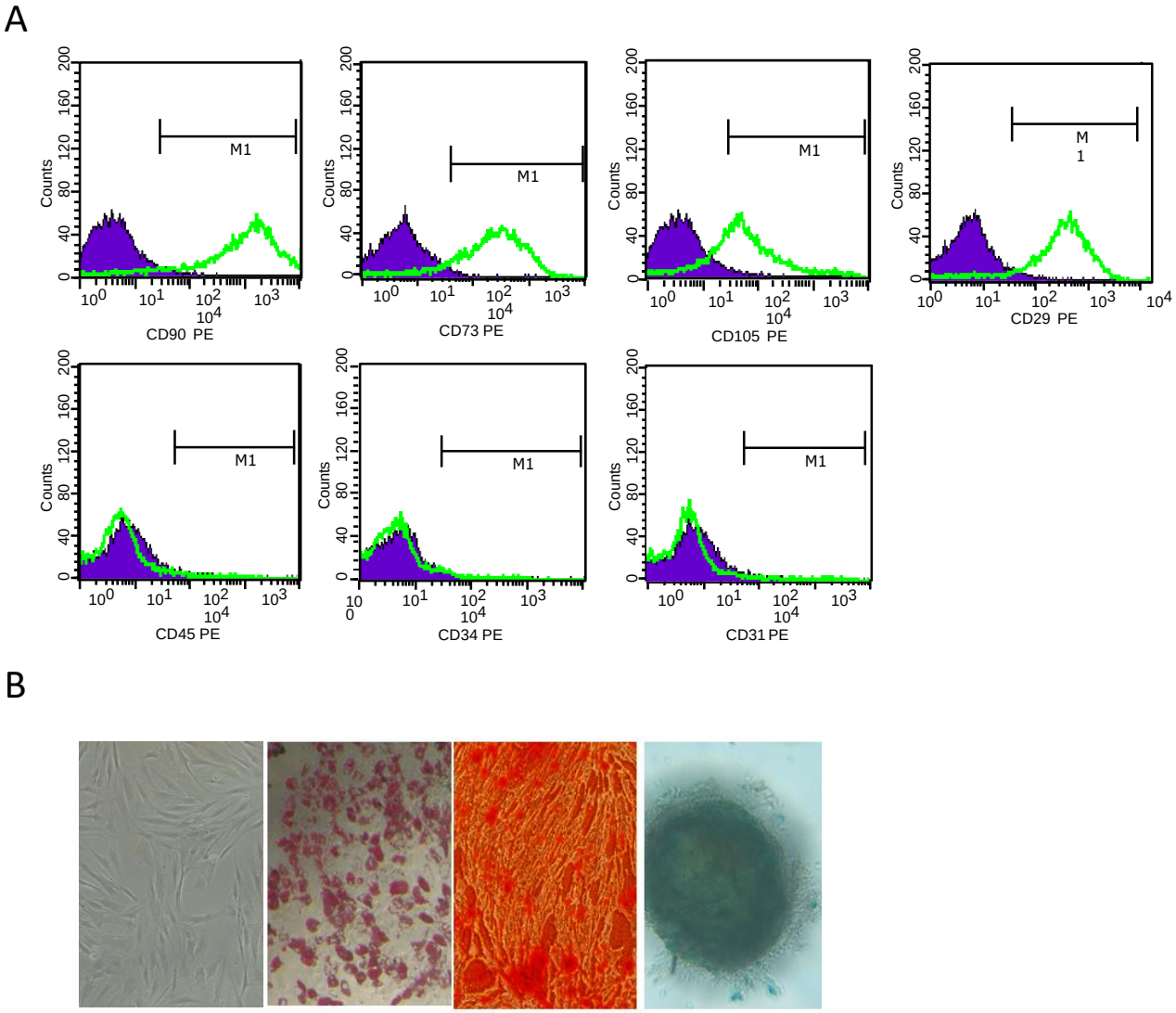

Figure S2. Characterization of ADSC phenotype and differentiation potential. (A) ADSC were stained with indicated antibodies and analyzed by flow cytometry. Histograms are representative of 5 independent experiments. Green lines represent stained cells and purple filled histograms their isotype-matched control. **(B)** ADSC were cultured in the presence of adipogenic, osteogenic or chondrogenic media for 21 days and then stained with either Oil Red O (middle left panel), Alizarin Red (middle right panel) and Alcian Blue (right panel) respectively. ADSC cultured alone served as a negative control (left panel) (magnification x10)

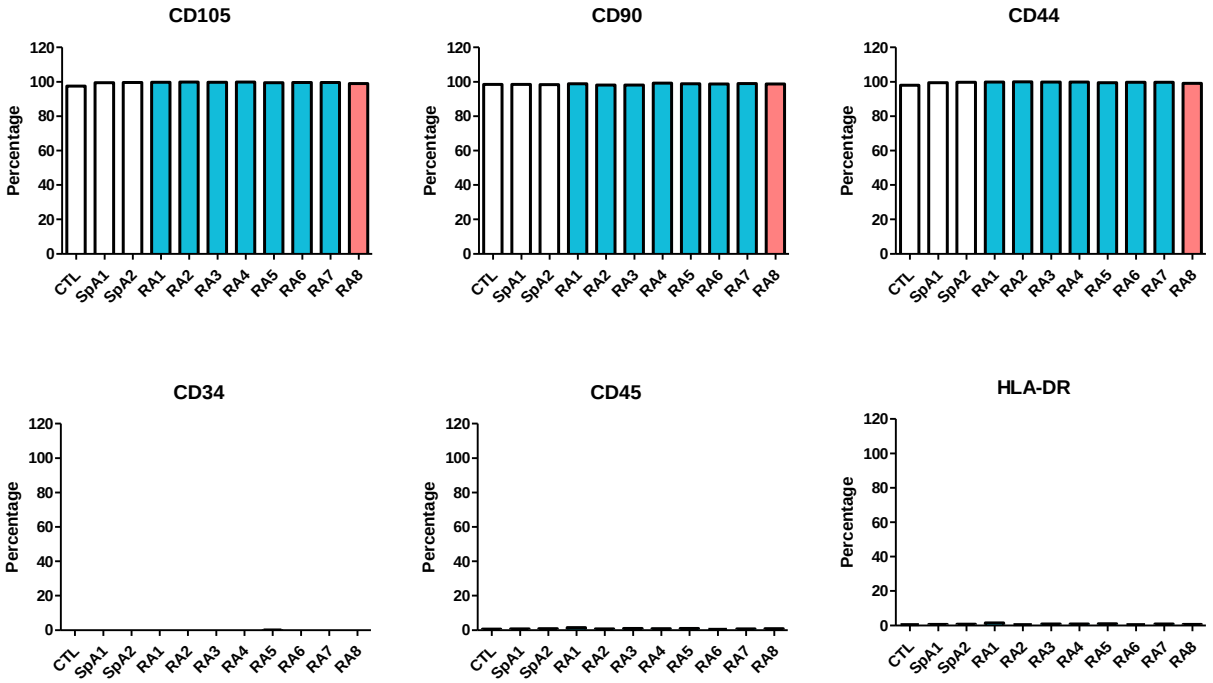

Figure S3. The phenotype of ADSC is not altered after SF treatment. ADSC were cultured for 24 hours in the presence of SF from either RA, SpA or CTL patients. Cells were then harvested and stained with anti-CD105, anti-CD90, anti-CD44, anti-CD34, anti-CD45 and anti-HLA-DR for their flow cytometry immunophenotyping.

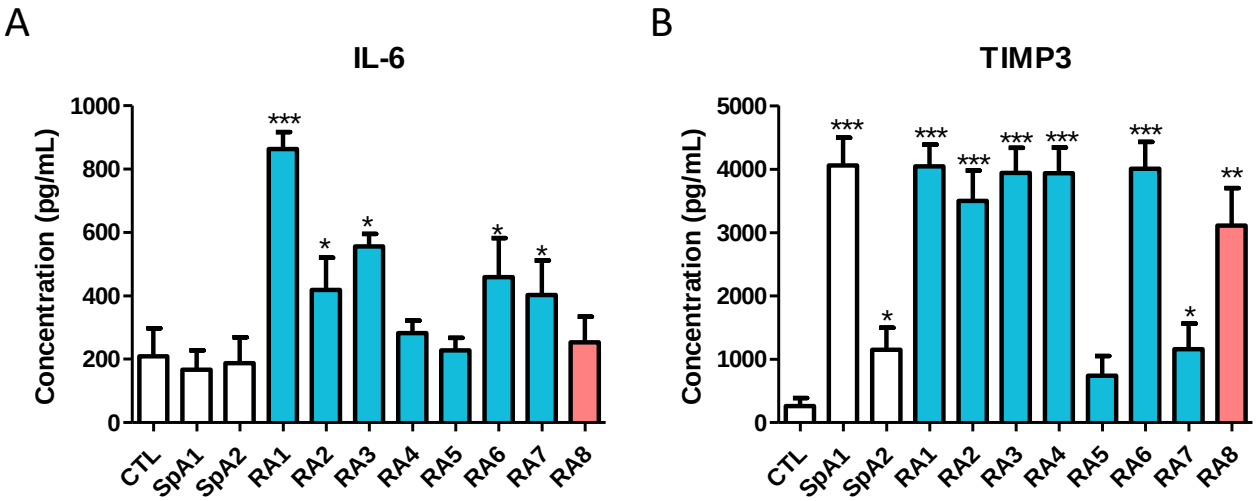

Figure S4. Differential effect of synovial fluids on ADSC secretion of IL-6 and TIMP3. ADSC were cultured for 24 hours in the presence of SF from either RA, SpA or CTL patients. **(A)** IL-6 and **(B)** TIMP3 were quantified in ADSC supernatants by ELISA. Results are represented as mean ± SEM of 5 independent experiments. *: $p<0.05$; **: $p<0.01$; ***: $p<0.001$.

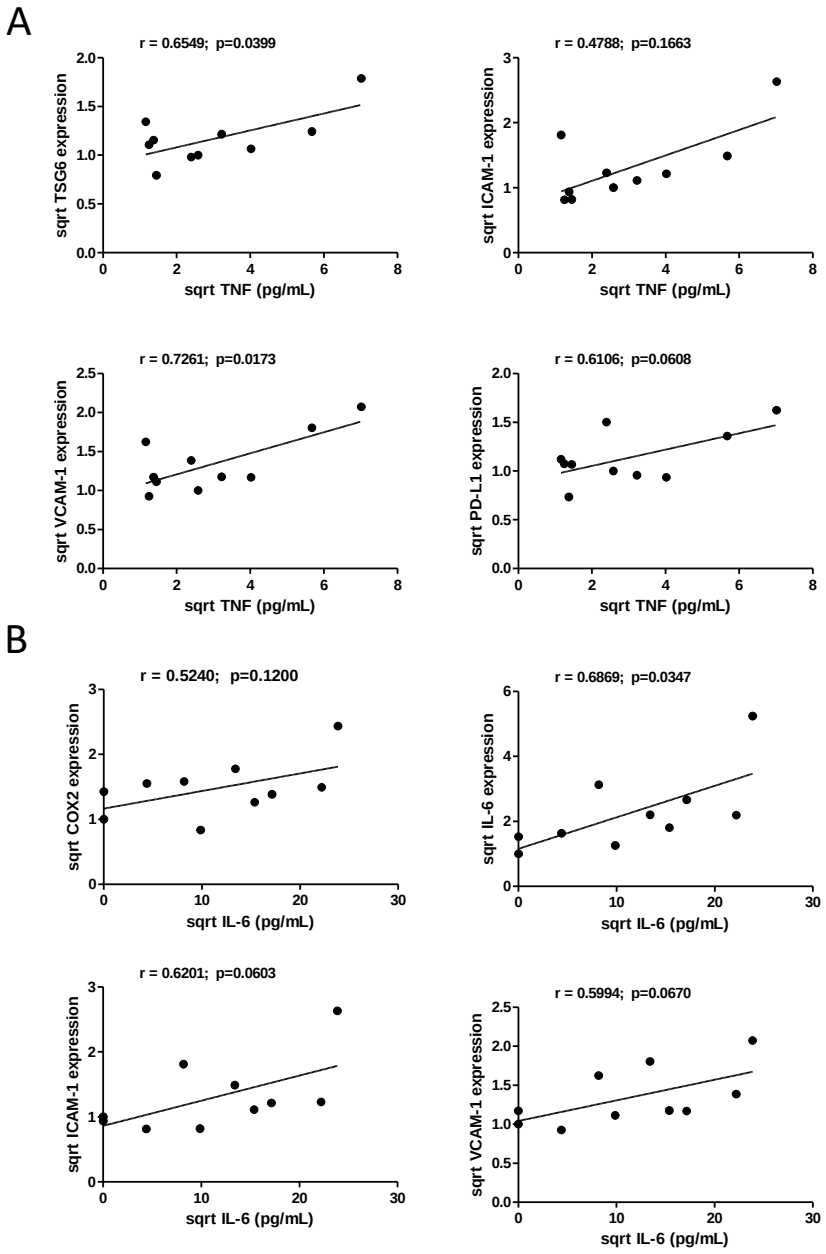

Figure S5. Correlation between TNF and IL-6 SF concentrations and ADSC gene expression. **(A)** Correlation (Pearson) between TNF concentrations and TSG6, ICAM-1, VCAM-1 and PD-L1 ADSC gene expression. **(B)** Correlation (Pearson) between IL-6 concentrations and COX-2, IL-6, ICAM-1 and VCAM-1 ADSC gene expression.

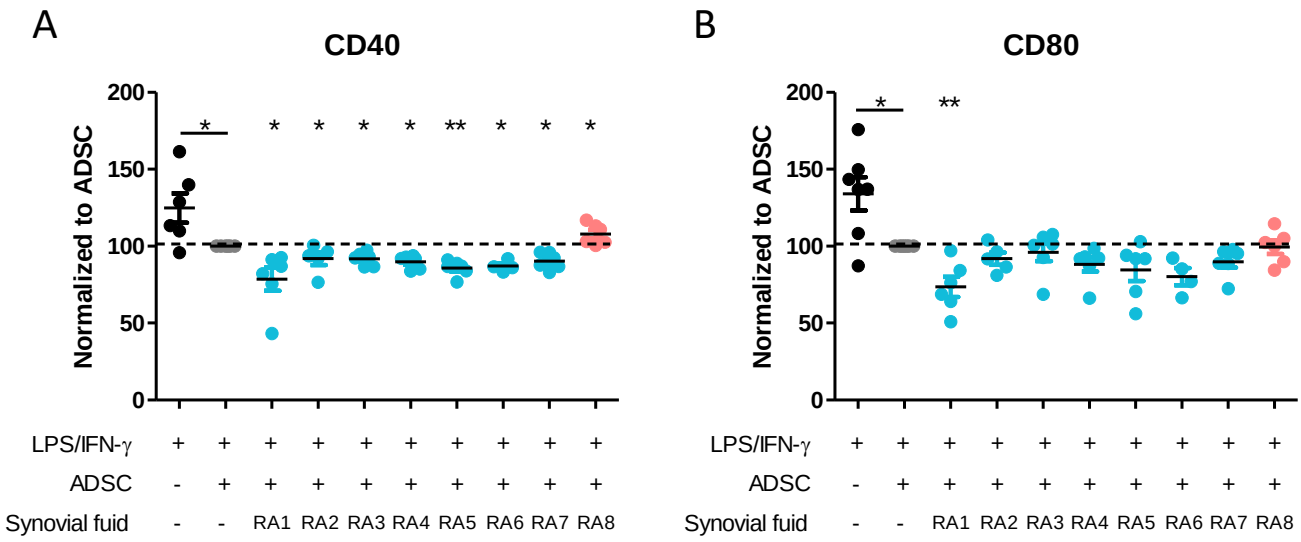

Figure S6. Conditioning ADSC with pro-inflammatory RASF enhances their ability to inhibit CD40 and CD80 expression in macrophages. ADSC were plated in 48-well plates and stimulated for 24 hours with RA1 to RA8. The following day, activated macrophages from healthy donors were added to ADSC at a ratio of 1:5 for 24 hours. Cells were then harvested and stained with anti-CD40 **(A)** and anti-CD80 **(B)** for flow cytometry detection of pro-inflammatory markers in macrophages. Results are represented as mean ± SEM of 4-7 independent experiments. Activated macrophages cultured in the presence of ADSC conditioned with different RASF were compared to those cultured in the presence of ADSC control. *: $p<0.05$; **: $p<0.01$.

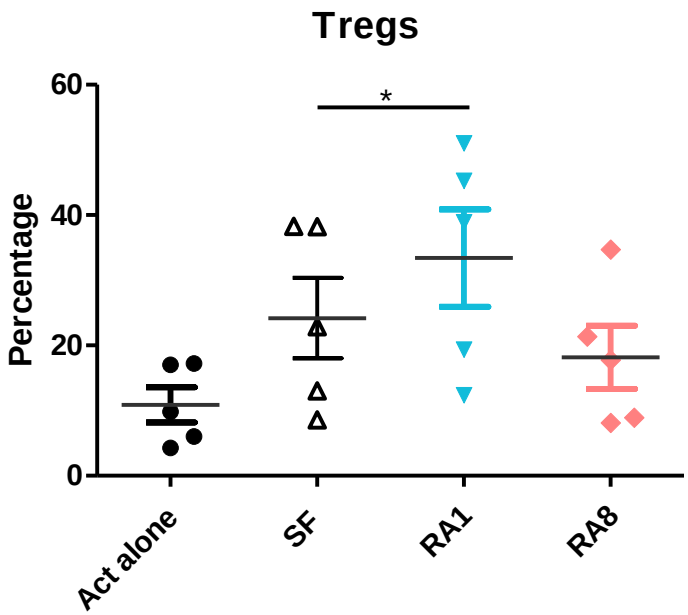

Figure S7. Conditioning ADSC with pro-inflammatory RASF enhances their ability to induce Tregs in PBMC. ADSC were plated in 96-well plates and stimulated for 24 hours with SF control, RA1 or RA8. The following day, ADSC were washed and PBMC cells from healthy donors were added to ADSC at a ratio of 1:5 and activated with beads coated with anti-CD3/CD28 for 72 hours. Cells were harvested and stained with anti-CD4, anti-CD25 and anti-Foxp3 for flow cytometry detection of Tregs. Results are represented as mean ± SEM of 5 independent experiments. *: $p<0.05$.

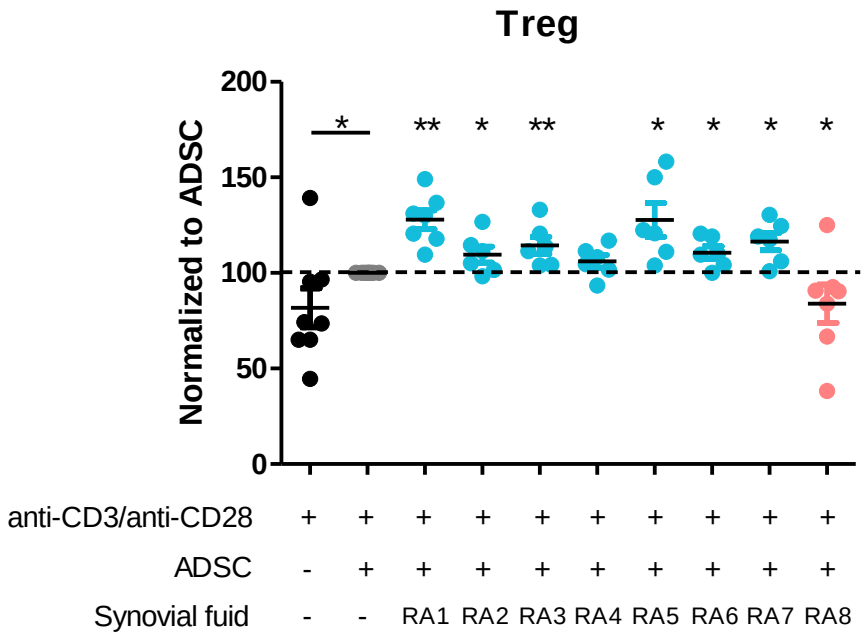

Figure S8. Conditioning ADSC with pro-inflammatory RASF enhances their ability induce Tregs. ADSC were plated in 96-well plates and stimulated for 24 hours with RA1 to RA8. The following day, ADSC were washed and T cells from healthy donors were added to ADSC at a ratio of 1:5 and activated with beads coated with anti-CD3/CD28 for 72 hours. Cells were then harvested and stained with anti-CD4, anti-CD25 and anti-Foxp3 for flow cytometry detection of Tregs. T cells activated in the presence of ADSC conditioned with different RASF were compared to those activated in the presence of ADSC control. Results are represented as mean ± SEM of 6-7 independent experiments. *: $p<0.05$; **: $p<0.01$.